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AEROMEDICAL REVIEWS

CARDIOPULMONARY RESUSCITATION

Review 10-62

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PART I: EVALUATION OF TEACHING PROGRAM

PART II: DIDACTIC PRESENTATION OF PROGRAM

CARDIOPULMONARY RESUSCITATION

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CARDIOPULMONARY RESUSCITATION

Part I: Evaluation of Teaching Program

Early in 1960, by diréction of the Surgeon General, United States Air Force, a course in cardiopulmonary resuscitation was organized and presented for physicians, dentists, and nurses assigned to the Medical Service, United States Air Force. The course was designed to teach these professional personnel the need for instruction in cardiopulmonary resuscitation and to give them the background information needed to develop courses at their home bases. Concurrent with this course, modified programs in pulmonary resuscitation were presented to the medical personnel of the USAF Hospital Lackland, permanent party personnel assigned to Lackland Air Force Base, and to other civilians in the San Antonio area. Between 1960 and 1961, more than 30,000 laymen were given instruction in pulmonary resuscitation.

Need for the course

The need for widespread instruction in cardiopulmonary resuscitation has long been recognized and was reiterated in the recommendations of the Symposium of Emergency Resuscitation (13) held in Stavanger, Norway, on 21 to 24 August 1961. One of the recommendations of this symposium was that "First-aid workers of all categories, school children, and the general public should be taught mouth-to-mouth and mouth-to-nose resuscitation." It was pointed out that the Norwegian government has already made it mandatory that all school children between the ages of 12 and 14 be given instruction in mouth-to-mouth and mouth-to-nose artificial respiration. This is a part of their regular school curriculum.

In an address given to the Ninth Annual National Conference on Disaster Medical Care, Brigadier General Don S. Wenger (48)

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pointed out that, in a mass casualty situation precipitated by a nuclear attack, physicians will not be immediately available; or if functional, they will not be at the right place, nor will it be possible for the patients to be brought to them. He emphasized the fact that during recent wartime experiences, the greatest casualty losses occured on the field prior to the time that the wounded were brought under the supervision of physicians. He also pointed out that hemorrhage and ineffective breathing efforts were the early killers. Thus, the injured themselves and the laymen who are in the area and fortunate enough to escape injury will carry the responsibility of instituting the treatment necessary to keep a large number of casualties alive for the hours, perhaps days, before they can be delivered into the hands of a physician. Respiratory resuscitation would undoubtedly be required in a large percentage of these cases.

Aside from a consideration of the first-aid requirements during a mass casualty situation, we are also currently concerned with the day-to-day happenings in which adequate respiratory resuscitation would play a significant role in salvaging lives. The incidence of death from asphyxia has been given by Safar et al. (42) as more than 20,000 people per year in the United States. Among the causes are drowning, electric shock, drug poisoning, carbon monoxide inhalation, chest injury, cardiac arrest, aspiration of foreign matter, and upper airway obstruction secondary to unconsciousness. For 1960, the World Almanac (51) gives the incidence of death from motor vehicle accidents as 35,820, from other accidents as 54,030, and from suicide as 18,330. Flagg (15) lists other causes of death not mentioned by Safar, including poisoning from industrial gases, smoke, and chemical warfare agents, acute pulmonary disease, anesthesia overdosage, high altitude flying, strangulation and suffocation, allergy, poliomyelitis, external pressure on the chest, and being trapped in caves or mines. In addition, the World Almanac gives an incidence of death during the perinatal period as 30,120. Summing the figures from the World Almanac, there occurred in 1960 a total of 137,800 deaths in which inadequate respiratory exchange assuredly played a vital role in the eventual terminal event, death.

In addition to the deaths from respiratory complications, the World Almanac lists 476,980 deaths from arteriosclerotic heart

disease including coronary disease. With the use of mouth-to-mouth resuscitation and external cardiac massage, extended life may be afforded many patients suffering these conditions.

Outline of course

The course in cardiopulmonary resuscitation held at USAF Hospital Lackland since 1960 is a one-day course of 8 hours. The following order of presentation has been found to be the most effective. Part II of this Aeromedical Review expands each item of the outline and. in essence, is the didactic portion of the course.

I. First hour
    A. Introduction
    B. Air Force Film FTA 458 (62)
    C. Purpose of the course

II. Second hour
    A. Normal respiratory physiology
    B. Pathophysiology secondary to airway problems
    C. Resuscitative technics
        1. Manual methods
        2. Direct artificial respiration

III. Third hour
    A. Mouth-to-mouth and mouth-to-nose resuscitation
    B. Role of drugs in resuscitation
    C. Army film TF8-3021 (55)

IV. Fourth hour
    A. Resuscitation of the newborn, film (58)
    B. Practice rescue breathing

V. Fifth hour
    A. Lunch
    B. Practice rescue breathing

VI. Sixth hour
    A. Introduction to cardiac resuscitation
    B. Factors precipitating cardiac arrest
    C. Prophylaxis of cardiac arrest
    D. Diagnosis of cardiac arrest
    E. Closed-chest manual systole

VII. Seventh hour
   A. Open-chest manual systole
   B. Comparison between open- and closed-chest manual systole
   C. Prognosis following cardiac arrest
   D. Closed-chest manual systole, film (57)

VIII. Eighth hour
   A. Practice endotracheal intubation on the dog
   B. Practice open-chest manual systole on an anesthetized dog
   C. Demonstration of defibrillation with the chest open
   D. Practice inflating the dog lung with the chest open
   E. Practice inflating the dog lung with the chest closed
   F. Practice closed-chest manual systole of an anesthetized dog
   G. Demonstration of defibrillation with the chest closed
   H. Questions and answers

Practical applications

Following the didactic portion of the course, a laboratory exercise is performed. Each member of the class is required to perform internal (fig. 1) and external (fig. 2) manual systole on a dog heart in ventricular fibrillation. In each instance a carotid artery is cannulated and connected to a calibrated mercury manometer. Each student is instructed, by demonstration, as to the most effective means of achieving an adequate systolic pressure with a resultant palpable femoral pulse. In addition, each member of the class is required to intubate the trachea of an anesthetized dog and to inflate the lungs of an anesthetized dog with the use of an anesthesia machine. Following the demonstration of manual systole, the use of various drugs and an electrical defibrillator are demonstrated by accomplishing internal and external defibrillation of the heart.

Discussion

Experience has shown that no single teaching medium can be relied upon to adequately instruct students in the technics of expired air resuscitation. All available teaching aids—films, airway demonstrators, and manikins (29)—as well as didactic presentations must be utilized. Even then a small percentage, approximately 1 or 2% in our experience, will never master an adequate technic of mouth-to-mouth or mouth-to-nose resuscitation. Even

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FIGURE 1

Laboratory preparation demonstrating the open-chest, positive-pressure respiration with oxygen delivered from an anesthesia machine, and a mercury manometer connected to the carotid artery for direct arterial pressure measurements.

though a student may be able to accomplish a resuscitative technic while under instruction, there is no assurance that he will remember this under the stress of an emergency. It is therefore vital that the simplest technic be taught and this, in the case of respiratory resuscitation, is also the most natural technic—the "head-tilt method" (12, 44).

In teaching the head-tilt method on a manikin, we have found that the technic is more readily learned if it is taught in a stepwise manner; that is, the student is first taught to achieve an airtight mouth-to-mouth and cheek-to-nose contact while the instructor

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FIGURE 2

Laboratory preparation demonstrating the closed chest, the mercury manometer, a recording apparatus for ECG determinations, and an external-internal electrical defibrillator.

maintains the head-tilt position. Once the student has learned how to keep a good mouth-to-mouth contact, he is taught to combine it with maintenance of the airway by tilting the head back.

Since the majority of the medical personnel attending the course will not be working in an operating room, the technic of closed-chest manual systole is given emphasis during the course. Practice with a viable physiologic preparation is invaluable since no mechanical apparatus adequately demonstrates the various problems which develop in the course of treatment. This applies to both the open- and the closed-chest technics of manual systole. The occurrence of complications can be demonstrated more readily under these circumstances and are more likely to be remembered.

We cannot answer the question as to what limitations should be placed on the use of the closed-chest technic, nor to whom the

technic should be taught. We have limited the teaching of the technic to medical, paramedical (hospital corpsmen), and qualified lifesavers (fire and rescue personnel and lifeguards). Until a study has been made of the use of closed-chest manual systole by the qualified lifesavers, we do not think it wise to advise a widespread use of the technic by other laymen.

Evaluation of results

An appraisal of the results of 12 lecture demonstrations given to physicians, dentists, and nurses from September 1960 to September 1961 was conducted early in 1962. Only those who were stationed in hospitals other than the USAF Hospital Lackland were surveyed. Among 135 people sent questionnaires, 112 replied. Each person was queried as to the number of lectures he had given to hospital personnel and to nonhospital personnel, and the number of attempts at cardiac or pulmonary resuscitation in which he had participated. Of those completing the questionnaire, 37 answered "no" or "none" to all of the questions. A summary of the positive results is given in table I.

TABLE I

Summary of number of lectures given by and resuscitative attempts by 112 former students in cardiopulmonary resuscitation

Category of lecturer	Lectures to—				Attempted resuscitations	
	Medical personnel		Laymen		Cardiac	Respiratory
	Number of lectures	Attendance	Number of lectures	Attendance		
Physicians and dentists	107	1,816	86	8,756	38	44
Nurses	30	484	3	72	7	14
Total	137	2,300	89	8,828	45	58

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It is encouraging to note that 11,128 people had been given further instruction by those attending the course, and it is hoped that the individuals now giving instruction will continue to present courses at their hospitals and duty stations. Only by a continued concerted effort by those trained in the technics of cardiopulmonary resuscitation can we ever achieve preparedness for a reasonable survival in the event of a widespread disaster. The incidence of cardiopulmonary emergencies is surprisingly high, as revealed by the survey, further indicating the need for preparation by medical personnel in cardiopulmonary resuscitative technics.

Part II: Didactic Presentation of Program

Respiratory resuscitation

Since much of the current literature pertaining to respiratory resuscitation cites specific values for some of the respiratory parameters, a brief review of the normal physiology will make these values more meaningful. The normal values of the respiratory gases are (1):

		Percent	*Tension (mm. Hg)*
Inspired air	Oxygen	20.94	158.25
	Carbon dioxide	0.04	0.8
Expired air	Oxygen	16.8	116.0
	Carbon dioxide	4.0	28.0

The factors which cause a change from the normal include: (a) asphyxia, which is defined as a lack of oxygen and an excess of carbon dioxide; (b) hypoxia, which is defined as a deficiency in oxygen (anoxia being a total lack of oxygen) and which classically is further subdivided into four types—hypoxic hypoxia, anemic hypoxia, stagnant hypoxia, and histotoxic hypoxia; (c) hypercarbia, which is defined as an excess of carbon dioxide (1). So far as pulmonary function is concerned, marked diminution in tidal volume (amount of air moved into or out of lungs during a single respiratory cycle—500 cc. normally), frequency (12 times a minute

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normally), or minute volume (amount of air breathed into or out of the lungs in one minute—6,000 cc. normally) (7) will result in the development of hypoxia and hypercarbia. Normal respiration is an automatic function regulated by the medullary centers in the brain stem. These centers are controlled by the carbon dioxide level in the blood; excess carbon dioxide results initially in an increase in both the tidal volume and the frequency of respiration. A less efficient and less critical control of respiration with normal breathing is available in the chemoreceptors located in the aortic arch and near the bifurcation of the common carotid artery. These areas are primarily influenced by hypoxia, and perform an important function in saving life by causing "terminal gasps" in severe laryngospasm. With an intact circulation and with breathing air, sudden cessation of respiration will result in a drop in the blood oxygen saturation from 95.8 to 76.3% within 60 seconds (47).

The pathophysiologic results of hypoxia or hypercarbia in relation to cardiopulmonary resuscitation cannot be considered as isolated occurrences because they develop simultaneously and influence each other. Excess of carbon dioxide causes local vasodilatation, increased muscular tone, headache, nausea, vertigo, unconsciousness, anesthesia, twitching, and convulsions. In the presence of hypoxia, a metabolic acidosis ensues, lowering the blood pH and the actual carbon dioxide content but still maintaining a high carbon dioxide tension. Hypoxia also causes many of the metabolic functions to fail with the eventual destruction of tissue. Initially, in most areas of the body, cellular metabolism continues through the anaerobic phase of glycolysis with the resultant metabolic acidosis previously mentioned. The structural elements of the central nervous system are subject to anoxic damage in a fixed order of vulnerability (26): (a) the neuron, (b) the oligodendroglia, (c) the astroglia, (d) the microglia, and (e) the connective tissue structures. In addition, it appears that the functional areas of the central nervous system are destroyed according to a selective pattern that is dependent on the nature of the hypoxia (i.e., whether it is the stagnant type or the hypoxic type). The actual pathologic effects of hypoxia on nervous tissue are of two types: (a) selective neuronal necrosis and (b) malacia.

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In May 1938, the First Conference of the Society for the Prevention of Asphyxial Death, Inc., now known as the National Resuscitation Society, Inc., studied the overall problem of asphyxia and its treatment; their study gave the problem widespread publicity. The first report on the failure of manual methods of respiration was made in 1958 by Safar et al. (35, 41). More recently the methods of artificial respiration were reviewed by Gordon et al. (16), Elam et al. (10), and Safar (34) in independent studies supported by the Army and the National Institutes of Health. In reports to the Council on Medical Physics of the American Medical Association, they concluded that mouth-to-mouth resuscitation is the most effective method available and the simplest and easiest to teach (8).

The manual methods of artificial respiration studied included the arm-lift, back-pressure (Holger-Nielsen) technic; the hip-lift, back-pressure technic; the hip-roll, back-pressure technic; the arm-lift, chest-pressure (Silvester) technic; the chest-pressure (Howard) technic; the prone-pressure (Schafer) technic; and the Eve rocking technic (16). None of these manual methods of resuscitation compared favorably with the mouth-to-mouth technic when subjected to laboratory measurement of blood oxygenation and tidal exchange. The key to the "new look" of emergency artificial respiration was the recognition of the role of airway obstruction, particularly pharyngeal soft tissue obstruction due to malpositioning of the head (34, 35, 41).

The most effective technic of respiratory resuscitation, mouth-to-mouth, is not new, for references to it can be found in the Bible: "And the Lord God formed man of the dust of the ground, and breathed into his nostrils the breath of life" (Genesis 2:7); "And he [Elisha] went up, and lay upon the child, and put his mouth upon his mouth," until finally the child who had seemed dead "opened his eyes" (II Kings 4:34-35).

It is obvious that some problem must be inherent in the technic of mouth-to-mouth resuscitation or it would have gained widespread popularity sooner. Undoubtedly, the esthetic distaste of mouth-to-mouth contact with an unknown victim has been a

FIGURE 3

Oral resuscitation devices.

primary factor in delaying its acceptance. This esthetic distaste
has led to the development of many gadgets to circumvent direct
mouth-to-mouth contact. Examples would include the Resuscitube
(Safar airway) (fig. 3) (33), the Ventibreather (28), the Brook
airway (3), the Mattick airway (31), anesthesia or plastic masks
(11), and resuscitator kits (45). Other devices (fig. 4), previously
introduced and advised for use in respiratory emergencies, were the
Kreiselman bellows (27), the Ruben bag and mask (82), and speci-
fically for infants the GBL (Goddard-Bennett-Lovelace) resusci-
tator (50). In addition to the above-named devices, a multitude
of mechanical resuscitators have been placed on the market as well
as IPPB (intermittent positive-pressure breathing) apparatus de-
signed for other purposes, but which can be adapted for use in
resuscitation. We recommend the use of a bag and mask of the
Ruben type (82) in emergency areas of the hospital where resusci-
tation might be required. This type of device has the advantage
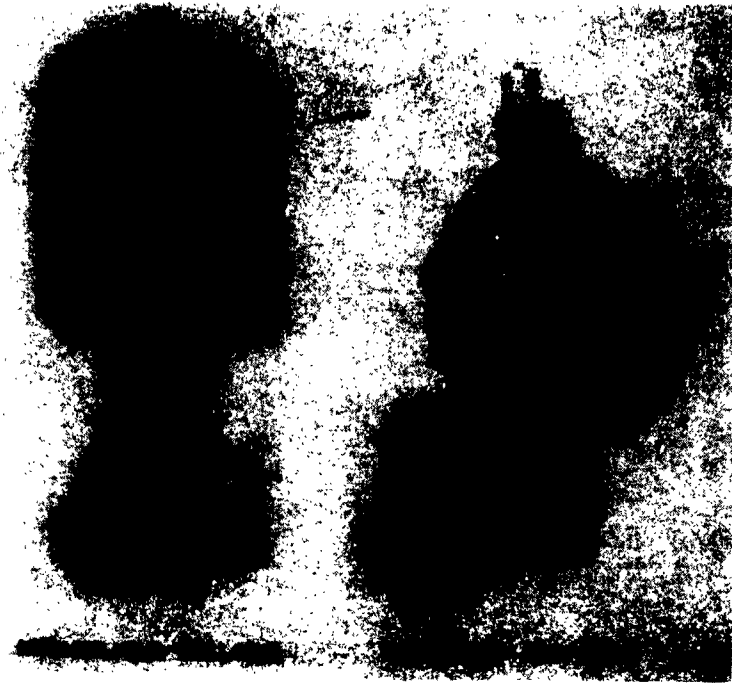that a high concentration of oxygen may be given under positive

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FIGURE 4

Hand-operated resuscitation devices.

pressure. Flagg (15) has given the most complete review of devices used for artificial respiration up to 1944.

Although many of the mechanical devices (named above) were designed to aid the rescuer in his attempts at artificial respiration, either by improving the adequacy of the airway or by obtaining an airtight fit over the mouth and nose, experience has shown that each device has some drawback when used by a layman (6) or even by a physician who has not been specifically trained in its use. The conclusion is that, for purposes of mass training, the only satisfactory technic that can be readily learned by a majority of trainees is the mouth-to-mouth technic. Experience has shown that simplicity of treatment is important to teaching any first-aid procedure (48). For this reason we advocate teaching the technic of mouth-to-mouth resuscitation, advising the use of the head-tilt

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method (12, 44) since in our experience this is the technic most readily learned by our trainees. To permit a certain amount of flexibility in the choice of technic, especially when mouth-to-mouth resuscitation is not feasible (i.e., in the case of the "tight-jaw"), we also require that our trainees learn a workable technic of mouth-to-nose resuscitation. Alternate technics such as the jaw-lift and chin-lift method (12, 44) are demonstrated to medical and paramedical personnel, and their use is permitted if the trainee is unable to adequately perform the preferred method. Actually, the greatest emphasis is placed on obtaining full tilting back of the head with all technics.

A word of caution must be given in any discussion of the mouth-to-mouth technic of resuscitation. The indiscriminate application of this technic can result in the spread of such infectious diseases as tuberculosis and meningitis. This possibility should not be used as an excuse for condemning the procedure; medical personnel should be aware of the possibility and should attempt to identify the disease in the victim and then treat the rescuer as indicated. If the diagnosis is known before resuscitation is required, it would certainly be preferable to use a mechanical device such as the Brook airway (8) or the Ventibreather (28), both of which are designed to prevent passage of the victim's expired air through the mouthpiece used by the rescuer.

It must be remembered that any technic of artificial respiration must produce a tidal volume of at least 500 cc. in the patient. None of the older manual technics can do this consistently in anyone, but mouth-to-mouth resuscitation can produce a tidal volume of 1,000 cc. or more in most patients. Even though expired air normally contains only 16.3% oxygen, anyone who does mouth-to-mouth resuscitation quickly realizes that he must breathe deeply to make the patient's chest rise, and in so doing he automatically increases the expired air oxygen concentration to about 18% (10). Concurrently with the increased tidal volume of the rescuer, the exhaled carbon dioxide percentage may not change or may rise to 4.5% because of increased metabolism (10). This small amount of carbon dioxide does not cause any significant increase above normal in the carbon dioxide content of the blood in the patient, and with

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a tidal volume close to 1,000 cc., more than an adequate amount of oxygen is supplied.

The use of teaching aids is vital to successful instruction in mouth-to-mouth resuscitation. Several excellent films are available (54-56, 59, 60, 62) and should be used to demonstrate resuscitative technics. Didactic portions of the instructive course should emphasize the need for: (a) immediate action, (b) inspection of the mouth for foreign material, (c) accomplishing full extension of the head on the neck—the "sniffing position," (d) the rescuer to open his mouth widely, (e) watching the rise of the chest, (f) the use of small puffs of air in children, (g) listening for the egress of air through the mouth, (h) watching the stomach for distention, and (i) compression over the stomach to prevent distention or to press out the air.

Whenever possible, a manikin (fig. 5) should be used for actual practice of mouth-to-mouth and mouth-to-nose resuscitation. Such manikins and airway demonstrators (fig. 6) are currently available (8, 29) and, although nothing is as good as practice on a human being, the devices are useful in learning the methods. In our experience, and in the experience of others (12), only a small percentage of students, 10 to 15%, can immediately develop adequate

FIGURE 5

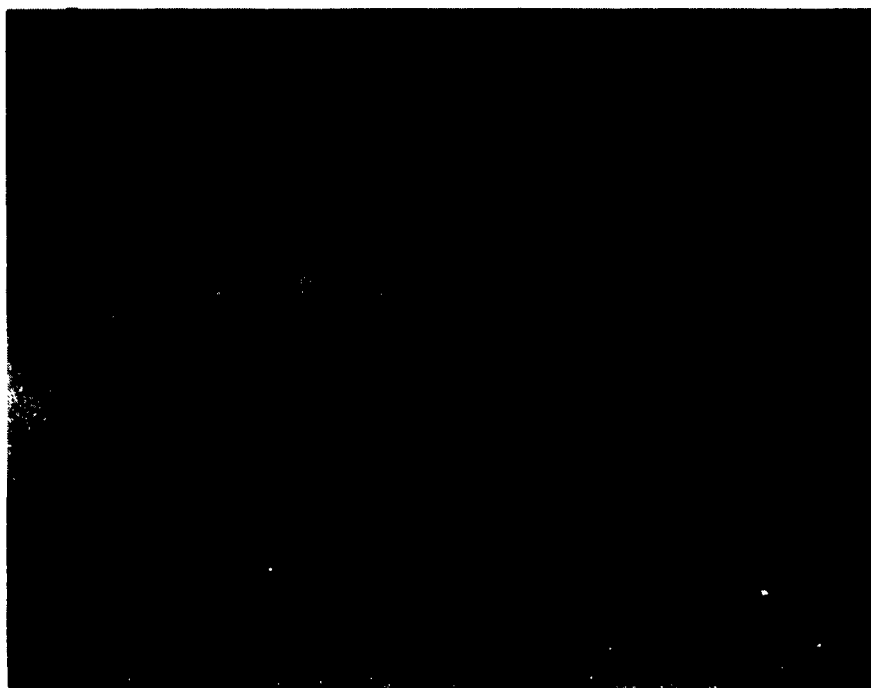*Resusci-Annie, resuscitation manikin.*

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FIGURE 6

Airway demonstrator.

respiratory exchange with mouth-to-mouth resuscitation after hearing lectures and seeing film demonstrations of the technic. On the average, it takes at least 5 minutes of practice on a manikin before a student can consistently move an adequate volume of air with the mouth-to-mouth technic. Even with the use of a manikin for practice, it will be found that a few students will never be able to move an adequate volume of air, but a persistent and patient instructor can reduce this percentage to 1 to 2%.

In our course, we advise against the use of analeptics in the treatment of respiratory depression. Specifically, we advise against the use of such drugs as nikethamide (Coramine), caffeine sodium

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benzoate, strychnine, amphetamine, alpha lobeline, pentamethyl-enetetrazol (Metrazol), methylphenidate (Ritalin), methylglutar-imide (Megimide), and brandy. Only if there is clear evidence of narcotic overdosage as a causative factor in respiratory depression do we advise the use of the specific narcotic antagonists. These drugs include levallorphan (Lorfan) and nalorphine (Nalline). It is stressed that when indicated, these drugs must be given intra-venously—levallorphan in a dosage of 1 to 2 mg. and nalorphine, 5 to 10 mg.

In discussing the specific problem of neonatal resuscitation, a film is available which explains the currently accepted philosophies and demonstrates very adequately the technics to be followed (58). Emphasis is placed on the necessity of preventing overdistention of the newborn lung with too vigorous an attempt at mouth-to-mouth resuscitation. It is pointed out that in small infants and children it is possible and preferable to do mouth-to-mouth and mouth-to-nose resuscitation simultaneously. If the lungs of the newborn are uninflated, the high pressures required for inflation by mouth-to-mouth respiration will invariably cause gastric dis-tention. Mouth-to-tracheal tube is therefore the technic of choice in newborn resuscitation. The problem of cardiac resuscitation in the newborn is considered briefly in the film. In electing the option of cardiac massage in the newborn infant, the physician must consider the length of time severe anoxia has been present and the possibility of the presence of congenital anomalies incom-patible with life. For instance, in the case of the stillborn infant, even considering the possibility that newborns are more resistant to the damaging effects of hypoxia (14), sufficient brain damage may have occurred by the time of delivery that the individual, if resuscitated, can serve no useful purpose to society. Certainly, in the event of a failing heart or cardiac arrest that can be readily documented, cardiac resuscitation should be attempted and prefer-entially, under these circumstances, by a closed-chest technic.

Cardiac resuscitation

The term *cardiac arrest* has been used for many years and has stood the test of time even though many attempts have been made

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to find a more exact word to describe the event. Whatever terminology is used, it is necessary to realize that we are not concerned with the physical inadequacy of the heart action alone, but that there is also a totally inadequate respiratory exchange. No attempt at cardiac resuscitation, by any technic, can be successful unless there is simultaneous respiratory resuscitation. In any discussion of the problem of cardiac arrest, we must concern ourselves with three facets of the problem—prophylaxis, diagnosis, and treatment. More recently we have available two forms of treatment, the older method of open-chest manual systole and the newer method of closed-chest manual systole.

As to the causative mechanisms of cardiac arrest, we should consider only those factors which are externally induced and eliminate the factors which normally occur in the pathophysiology of aging or in a disease process. The only exceptions to this would be in the event of a myocardial infarction, a pulmonary embolus, a cerebral embolus or thrombosis, and a Stokes-Adams attack. No matter what the specific event might have been which led to cardiac arrest, hypoxia is always the end point and the factor that prevents the heart from immediately recovering from the event. Resuscitative attempts should always be directed toward overcoming the hypoxia first, and then toward overcoming the precipitating factor. Once the hypoxia has been corrected, the human body will often adjust to the precipitating factor if it still exists, but under many circumstances this factor has ceased to exist.

Changes in coronary blood flow which result in an inadequate supply of blood and oxygen to the myocardium may be in the form of a diminished blood flow, or may be only relative because of an increased demand for oxygen by the myocardium. Hypotension from any cause will cause a diminished blood flow through the coronary arteries. This hypotension may be a result of hemorrhage, spinal, epidural, or local anesthesia, positioning, faulty placement of retractors, or torsion of the heart and great vessels. The coronary flow may be diminished from an overdosage of drugs or general anesthesia. It may also be caused by a raised airway pressure which prevents the normal return of blood into the thoracic cage, or by a simple traction reflex. An increase in circulating

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epinephrine, either endogenous or exogenous, will cause an increased demand for oxygen and, therefore, a relatively inadequate coronary flow.

On the other hand, the coronary blood flow may be relatively adequate, but the myocardium and the conductive mechanisms of the heart may suffer a direct depression. This depression may originate from a reflex—carotid sinus, pulmonary vagal, or hilar. The depression may result directly from drugs—narcotics, general anesthetic agents, cardiac glycosides, quinidine, or procaine amide.

Even with an adequate coronary blood flow and a normally functioning myocardium, the blood supplied to the heart from the lungs may be lacking in available oxygen. Any form of respiratory obstruction or respiratory depression will prevent the transfer of adequate amounts of oxygen into the blood. In addition, there are numerous miscellaneous factors which have a multiplicity of effects or acti᷾ ᴊ (e.g., electrocution, hypothermia, or direct cardiac manipulation during surgery). Again, it is pointed out that, regardless of the precipitating factor, hypoxia results, and immediate and often drastic remedial action is required.

Many of the factors precipitating cardiac arrest are iatrogenic. In this area we can improve in the prophylaxis of cardiac arrest. Up to about 1944 the reported incidence of what we now term cardiac arrest was relatively low. Undoubtedly the same precipitating factors were in existence prior to this time and cardiac arrest was occurring, but the diagnosis per se was not made. During the decade following, there was a considerable rise in the reported incidence of the diagnosis. This most certainly was a result of the greater emphasis placed on the diagnosis in the medical literature and of the favorable publicity the treatment received from the lay press. The disturbing fact is that 20 years after cardiac arrest was first recognized, the incidence of cardiac arrest is still on the increase. Because of the space given the subject in medical journals and its addition to the medical school curriculum, it can be assumed that the problem of cardiac arrest is well understood. Some have explained the continued rise in incidence as a result of the advances in medical knowledge and technics which have permitted us to operate on patients who previously were denied surgery because

of being too poor a risk. This assuredly is a partial explanation, but must not be taken as the final answer. Hewlett et al. (18) have introduced the term *cardiac arrest suspects*. In this category they place all patients with marked heart disease, toxemia, inanition, cardiac decompensation, chronic cyanosis, and toxic states as evidenced by long periods of temperature elevation, anemia, respiratory embarrassment, or chemical imbalance. For these patients a more concerted effort must be made to stabilize the pathologic processes and to bring the physiologic processes back to as near normal as possible before subjecting the patient to surgery. They feel that "the value of unhurried preparation far exceeds the most heroic emergency efforts."

A definition of cardiac arrest has been given as "The clinical picture of apnea, unconsciousness, and pulselessness in a person who had not been expected to die" (25). Many criteria for the diagnosis of cardiac arrest have been given. The most frequently mentioned criteria include: the absence of a radial or carotid pulse, inaudible heart sounds, sudden pallor or cyanosis, sudden pupillary dilatation, respiratory standstill or apneustic breathing, the absence of bleeding or the appearance of dark blood in the operative field, and electrocardiographic evidence of asystole or ventricular fibrillation. The most significant criterion is the absence of a pulse in a large artery, and this alone is sufficient to institute therapy. One must not wait for electrocardiographic confirmation, nor should one run through any list of signs for confirmation. Time is critical. Treatment must be started immediately. The medical literature cites periods of time ranging from 3 to 5 minutes after which anoxia will invariably produce some degree of irreversible damage to the central nervous system. When a chronic state of hypoxia has existed for a prolonged period, the anoxia developing after cardiac arrest will cause irreversible damage in something less than 3 minutes. When respiratory anoxia alone kills a healthy person, central nervous system function is impaired before the heart stops. Confirmation of the diagnosis, either by electrocardiogram with closed-chest manual systole or by direct vision with open-chest manual systole, may be made after treatment has been initiated.

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Closed-chest manual systole. The recent description by Kouwen-hoven and his associates (22, 24) of a technic of manual systole without opening the chest has caused many changes in the philosophy of the treatment of cardiac arrest. The value of this technic of "external cardiac massage" lies in its simplicity and adaptability. Once the lack of effective heart action has been ascertained by the lack of a palpable peripheral pulse or the lack of audible heart sounds, the patient is placed supine on a hard, flat surface and treatment is started. As is true with open-chest manual systole, manual systole and artificial respiration must be performed simultaneously (43). While one rescuer performs mouth-to-mouth resuscitation, another rescuer performs manual systole by exerting external pressure over the sternum. In performing manual systole on an adult patient, the heel of one hand is placed on the lower one-third of the body of the sternum, and the second hand is placed over the first and perpendicular to it. Care must be exercised that the lower hand remains above the xiphoid process of the sternum and medial to the costal cartilages. Rhythmic pressure is then exerted over the sternum at a rate of 40 to 50 times a minute. The sternum should be depressed 3 to 5 cm. when the pressure is applied. Pressure is maintained for approximately ½ second. This pressure forces blood out of the heart into the great vessels. With release of the pressure, the heart refills. Pulmonary inflation is timed to occur between compressions.

In the event that only one rescuer is available, both manual systole and mouth-to-mouth resuscitation may be performed by the same person. Various ratios of manual systole and pulmonary inflation have been recommended (4, 17, 36), but recent laboratory studies (40) have shown that severe deoxygenation of arterial blood occurs during 30 seconds of closed-chest manual systole without pulmonary ventilation, but not during 15 seconds of manual systole without ventilation. It is, therefore, recommended that 3 rapid lung inflations be alternated with 15 sternal compressions. This cycle can be used for both single operator resuscitation and dual operator resuscitation.

Thus it can be seen that no special equipment is required to perform resuscitation. On the street or the open ward of the hospital one need not hesitate because of the lack of a proper

surgical atmosphere. We have pointed out previously that the urgency of the emergency existing when the heart has stopped or is in ventricular fibrillation is one of the greatest, and a few seconds may make the difference between life and death. External manual systole may be instituted immediately and without hesitancy. The use of drugs can be the same as used with open-chest manual systole. Intracardiac injections are made through the fourth left interspace, close to the sternum. Simulated rapid transfusion may be accomplished simply by elevating the legs. This will have the same effect, temporarily, of transfusing 500 ml. of whole blood.

Electrical defibrillation may be accomplished externally with the use of an apparatus which will deliver 440 volts of electricity (52). The electrodes for external defibrillation should have 58 cm.^{2} of surface area and should be coated with electrode jelly. One electrode is placed over the apex of the heart and the second over the top of the sternum at the sternal notch, along the long axis of the heart. Firm pressure should be exerted on the electrodes to assure widespread contact with the skin. The current should be delivered for 0.25 second. Repetitive shocks are often necessary. Kouwenhoven is reported by Safar (38) to advise the use of external defibrillation if pulselessness persists after a few minutes of cardiac compression, even if an electrocardiogram is not available. He has shown that a shock of this type does nothing more to the normal heart than to achieve maximum contraction. Care must be taken that the shock is delivered along the long axis of the heart and not across the heart. Manual systole should precede defibrillation under all circumstances. When external defibrillation is used on infants or children, 220 volts for 0.25 second is usually sufficient.

The results obtained with the use of closed-chest manual systole are promising. There have been only a few circumstances reported (20, 36) in which this technic was ineffective and it was necessary to open the chest. Although the group at Johns Hopkins Hospital uses the closed-chest technic almost exclusively (39), even in the operating room, we do not agree. We feel that the risk of infection or trauma to the heart, great vessels, or lung in opening the chest in the operating room is less than the risk of trauma

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seen with the closed technic. We advise the use of closed-chest manual systole in all areas other than the operating room suite and the use of open-chest manual systole in the operating room.

Serious trauma may result from an improper application of external manual systole. Multiple rib fractures have been encountered (22) but are compatible with life unless a flail chest develops. The more serious complications include subcapsular liver hematoma or rupture of the liver (22, 37) and torn mediastinal vessels with extensive hemorrhage (22, 37). These complications may be incompatible with life. Special care must be exercised in the application of external manual systole to infants and children. In infants, the pressure from the thumb or several fingers of one hand may be sufficient to obtain adequate circulation of blood. In children, the pressure exerted by the heel of one hand is usually sufficient to achieve an adequate manual systole.

Because of the inherent risk of severe incapacitating trauma, and because one must exercise medical judgment in making the diagnosis of cardiac arrest, it has been recommended that the teaching of the closed-chest technic be limited, for the present, to medical personnel and recognized lifesavers (13). This implies careful instruction with appropriate warnings of the pitfalls in the use of the technic, especially when instructing personnel other than members of the medical profession.

Open-chest manual systole. Several films are available which demonstrate the approved technics of open-chest manual systole (53, 61), and many reviews have appeared in the literature (2, 5, 9, 30, 46, 49). We advise a 30-second trial of closed-chest manual systole prior to making the skin incision for open-chest manual systole. If a spontaneous palpable pulse has not returned by this time, a skin incision should be made in either the fourth or fifth left intercostal space with the patient lying supine. The incision should start just lateral to the border of the sternum and should extend laterally to the level of the operating table. In carrying the incision through the deeper tissues, care must be taken not to incise the internal mammary artery, lying just lateral to the border of the sternum, or the visceral pleura. The costal cartilages above and below the incision should be severed medially. The ribs may

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now be spread apart, the heart compressed and then observed. If it is in asystole or ventricular fibrillation, one hand must be inserted into the chest and manual systole started. Once the treatment has been started, there must be no delay—the skin is not prepared and surgical gloves need not be used. Once the chest is open and adequate manual systole begun, then the urgency is over and routine sterile precautions should be instituted.

Several different technics of accomplishing manual systole are possible. The heart may be pressed upward against the sternum, the heart may be held in one hand and compressed, or the heart may be compressed between both hands. For any technic to be considered adequate, a peripheral pulse, carotid or femoral, must be palpable with each manual systole. If this cannot be accomplished, then the pericardium should be opened. The pericardium should be incised swiftly from the base to the apex of the heart with care being taken that the phrenic nerve is not cut. Manual compression should again be started. The rate of compression should be about 60 times a minute, but will also be governed by the time it takes for the heart to refill. After each compression all pressure should be completely removed from the heart or there will be incomplete filling. During compression of the heart with one hand, the second to fifth fingers should be used as a flat surface to bring the heart against the palm. Care must be taken that the thumb or another finger does not penetrate an auricle or the right ventricle. If a rate faster than 80 times a minute is attempted, it will be found that the hand tires rapidly and effective massage cannot be maintained. If massage is continued longer than 5 minutes, a second person should perform the manual systole for the next 5 minutes since 5 minutes of manual systole is very tiring.

Concurrent with the start of treatment, artificial respiration must be given with 100% oxygen if it is available. Ventilation by mouth-to-mouth or by bag and mask should precede tracheal intubation. As soon as possible after the start of treatment, intravenous therapy should be instituted. The specific type of fluid to be used will depend upon what is immediately available. Usually, it is eventually necessary to resort to blood transfusions. Volume is essential early, but once the heart begins to refill readily, care

must be taken not to overload the circulation. If extra people are available, someone should record the timing of events.

The use of drugs in the treatment of asystole or ventricular fibrillation should be delayed until manual systole has been performed. Drugs may be helpful in changing an abnormal heart rhythm to a normal sinus rhythm or in strengthening the force of myocardial contraction. Various drugs and combinations of drugs have been proposed in the past (9, 22, 25, 30, 37, 46, 49, 53, 57). We recommend the use of 5 to 10 cc. of a 10% solution of calcium chloride injected into the right ventricle if the tone of the heart muscle is poor and if refill is poor. As soon as it is available, we almost routinely inject into the right ventricle 5 to 10 cc. of a mixture consisting of Isuprel 0.1 mg., phenylephrine 4 mg., and atropine 1 mg. q.s. to 20 cc. with normal saline. The solution is kept available in a bottle on the cardiac arrest cart. Two to three ml. of a 1/10,000 dilution of epinephrine is occasionally injected into the right ventricle. If there has been a prolonged period of manual systole, 5 gm. of sodium bicarbonate are given slowly, intravenously.

In the event that ventricular fibrillation has occurred, the same form of treatment is given as has been previously described for asystole. Only after adequate manual systole is accomplished should any form of defibrillation be attempted. When available, an electrical defibrillator developing 110 to 160 volts should be employed. The electrodes should be at least 7 cm. in diameter and should be wrapped in gauze soaked in normal saline. In placing the electrodes on the heart, care must be taken that a maximum mass of ventricular muscle is held between the electrodes. In the first attempt at defibrillation, only 110 volts for 0.1 second should be used. If this is ineffective, the heart should again be manually compressed before repetitive shocks are given. If repetitive shocks are ineffective then the voltage may be increased to 160 volts or the duration of the shock may be increased to 0.25 second, and a similar series of shocks given. Kaiser et al. (23) have shown a high incidence of severe myocardial burns in dogs with the use of 230 volts for 0.25 second and a moderate incidence of burns at 0.1 second. If possible, the higher voltages and durations should

be avoided. Once the heart has been defibrillated, it will be in asystole and usually will not resume spontaneous activity until manual massage has been given for a brief period of time. The drugs named earlier may be used. On occasion it may be necessary to use them before defibrillation can be successfully accomplished. If the heart is cold, defibrillation will be unsuccessful until the temperature is above 30° C. Warm saline poured into the open chest will rewarm the heart quite rapidly.

In the event that an electrical defibrillator is not available, potassium chloride may be used. Potassium chloride (6 to 8 mEq.) should be injected into the root of the aorta while the aorta immediately distal to this point is clamped shut. With the aorta still clamped shut, manual systole will force the drug into the coronary arteries. This will then put the heart into asystole and manual systole must be continued after unclamping the aorta until the drug is removed from the myocardium and a spontaneous beat occurs.

After a cardiac arrest has occurred, there always comes a time when the relatives of the victim must be informed of the prognosis. This is always difficult and statements must be guarded, but the observation of certain signs will aid considerably in making the prognosis. Certainly, if the heart responds very rapidly to the treatment, the pupils revert to a normal size after having been dilated for only a brief period of time, a normal respiratory pattern is quick to return, and the patient rapidly regains consciousness, the prognosis for an uneventful recovery is quite good. On the other hand, if none of these occur rapidly, if the patient develops convulsions, and if excessively high fever persists, the prognosis becomes very poor. The patient may recover only partially and exhibit multiple neurologic deficiences, or may die.

If cardiac arrest has occurred in the operating room before or shortly after surgery has been started, it is usually wise to try to postpone the surgery. The patient may be returned to the operating room in 10 to 14 days for the completion of his surgery if there was an immediate response to manual systole and signs of immediate recovery are apparent. If a moderate amount of manual systole

was required before resumption of adequate cardiac activity and if there has been only minimal asphyxia, the patient may be returned to the operating room in 2 to 4 weeks. If the response was slow, with prolonged asphyxia and evidence of myocardial damage, the patient should be treated as though he had had a severe myocardial infarction and should not be returned to the operating room for at least 3 months (19). Before the patient is returned to the operating room, the preoperative preparation must be done with thoroughness and care. Anesthetic management must be cautious, but no specific agent need be avoided if hypoxia and hypotension are prevented.

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